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CLINICAL PROCEEDINGS

of the

CHILDREN'S HOSPITAL

WASHINGTON, D. C.

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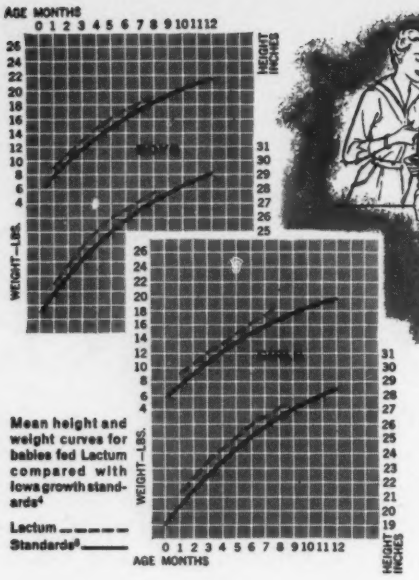

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OF THE CHILDREN'S HOSPITAL

13th and W Streets, Washington 9, D. C.

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PEDIATRIC GLAUCOMA

Thomas E. Haggerty, M.D.*

INTRODUCTION

Glaucoma may be simply defined as an increase in intraocular tension. Normally the intraocular tension is between 14 and 30 mm. mercury. An elevated tension alters the anatomy and physiology of the eye so that eventual blindness results. The anatomic alterations depend upon the stage of development of the eye at the onset of increased tension. If an eye is fully developed and matured, the increased pressure causes an excavation of the nerve head with the destruction of neural fibers as they enter the eye. If, however, the eye is immature and not fully developed, the increased pressure causes an enlargement of all the ocular coats, resulting in a large eye.

There are two terms used to denote this later condition of large eye: buphthalmos and hydrophthalmos. Recently it has been a practice to use both terms synonymously, but the pediatrician would do well to think of hydrophthalmos as resulting from a defective filtration angle and buphthalmos as resulting from any cause other than a defective angle. This differentiation gives clues to the pathology and etiology. In any case, these terms denote a glaucoma occurring during a phase of an immature eye.

Congenital glaucoma is a relatively rare disease, the incidence among normal children varying from 0.03 to 0.08%. However, it accounts for from 5 to 13% of blindness among children of school age, and thus has important social and economic implications. As will be shown, earlier diagnosis and improved treatment will lead to a reduction in the incidence of blindness. It is thus an important pediatric entity.

CLASSIFICATION

The following classification of pediatric glaucoma was devised to coincide with the commonly used pediatric classification of disease, that is, infantile and juvenile. This distinction is based on two concepts. In a limited sense it is based on age; but in a broader sense, it is based on whether the glaucoma was produced by congenital abnormalities, or whether it was acquired, i.e., produced by post-natal abnormalities. A classification made in this manner allows the pediatrician to orient his thinking in terms of the age group of the child.

1. Infantile (Congenital) Glaucoma: secondary, or closely related to, developmental anomalies
   A. Anterior attachment of the root of the iris

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B. Persistence of mesenchyme in the filtration angle
C. Partial or complete absence of Schlemm's Canal
D. Angiomatosis
E. Neurofibromatosis
F. Aniridia
G. Microcornea
H. Retrolental fibroplasia
I. Microphakia

2. Juvenile Glaucoma:
A. Following penetration of the cornea, or perforating corneal ulcer
B. Following massive hemorrhage into the posterior ocular segment
C. Associated with active or healed iridocyclitis, or cyclitis, or anterior choroiditis with blocked angles.
D. Associated with tumor growth, blocking angles.
E. Massive hemorrhage into the anterior chamber.
F. Adult primary glaucoma. Very rare below the age of 20 years.

EMBRYOLOGY

In order to obtain a clearer concept of the pathology and mechanism of congenital glaucoma, brief mention will be made of several steps in the embryological development of the trabecular—Schlemm's Canal mechanism. Aqueous humor, formed by the processes of the ciliary body, passes anterior to the lens, out through the pupillary opening into the anterior chamber, drains laterally into the angle of the anterior chamber, filters through the trabecular meshwork into the canal of Schlemm, and thence back into the general circulation by means of the aqueous and episcleral veins. A small amount of aqueous bypasses this route by filtering into the iris. That portion of the eye which develops into the anterior chamber is filled with a mesodermal meshwork from the 16 mm. stage. At about the end of the fifth month the anterior chamber begins to form. At the 65 mm. stage, a small venous channel appears in the fibers of the cornea-sclera. This later becomes the canal of Schlemm. After the fifth month, the mesenchymal tissue filling the anterior chamber starts to atrophy, and is fairly well cleared by the end of the seventh month. At the same time, the mesenchymal tissue between the angle and Schlemm's canal atrophies, so that only the trabecular meshwork is left at the end of the eighth month.[1]

It should be noted also that the root of the iris is inserted into the anterior surface of the ciliary body by connective tissue which passes into the substance of the ciliary body. In most cases of infantile glaucoma, the iris is found to be inserted anterior to the ciliary body, even as far forward as the cornea.

MECHANISM OF GLAUCOMA

Reference to the preceding classification shows that the principal mechanism causing an elevation of intraocular tension is obstruction; the site of the obstruction being the variable and distinguishing feature. There is some indirect evidence that excessive formation of aqueous humor may play a role in some of these conditions, but obstruction is the more frequent cause of glaucoma. This obstruction can be broken down as to location.

1) Obstruction in the angle: as for example in anterior attachment of the iris, persistence of mesenchyme in the angle, aniridia, microcornea, perforation of the cornea, retrolental fibroplasia, and posterior ocular hemorrhage. In this type of obstruction, contact between the aqueous and the trabecular spaces is prevented because of the presence of mesenchymal tissue in the angle, or because the iris is sealed over the trabecular spaces.

2) Obstruction in the trabecular spaces: due to cellular debris, connective tissue, or blood, as found in iridocyclitis, cyclitis, or hemorrhage into the anterior chamber.

3) Obstruction due to the congenital absence of Schlemm's Canal:

Glaucoma accompanying active cyclitis, or angioma of the ciliary body, results from the excessive secretion of aqueous.

There are two clinical entities which should be mentioned at this time because of their close association with glaucoma. There is a frequent association between extraocular angiomata and glaucoma. Capillary or cavernous hemangiomata, involving the distribution of the fifth cranial nerve, manifested as port-wine staining of the skin of the face, or telangiectases of the conjunctiva, sclera, or choroid, in an infant, should lead to a suspicion of glaucoma. The nevi may also involve the meninges, producing a Sturge-Weber Syndrome. Also, neurofibromatosis may be associated with glaucoma; which may occur early enough to produce buphthalmos, or may occur in adult years. The glaucoma in these cases results from associated anomalies, such as a poorly developed iris, corneo-iris adhesions, and infiltration of the uveal tract with the fibrous-like cells of Schwann.

CLINICAL PICTURE

Clinically, three distinct groups are observed, depending upon whether anatomic changes are appreciated when the child is first seen. Ocular hypertension is common to all groups. In the first group, the child is born with, or develops shortly after birth, enlargement of the corneal diameters, generalized enlargement of the globe, deepening of the anterior chamber, and clouding of the cornea. These findings may be present in varying proportions. But there is measurable anatomic alteration. As the eye stretches rapidly, the nutrition of the cornea is disturbed, and as the tension rises,

aqueous seeps and is forced into the corneal stroma, reducing its transparency. If this situation persists, corneal opacities are formed, and the cornea may present a stippled or striped appearance. As the tension rises, conjunctival and episcleral vascular engorgement are noted. In these cases, cupping of the disc does not occur until late in the course, since the cornea and sclera are more easily stretched than is the lamina cribrosa. Cataract formation also occurs late in the course. The prognosis is poor.

In the second group, there are no gross anatomic alterations. The eyes may appear to be symmetrical and of normal size. Of course, if the tension is determined instrumentally, it will be found to be elevated; and careful measurement of the corneal diameters may reveal a slight enlargement or asymmetry. Clinically, children in this group present a picture of chronic or recurrent lacrimation and photophobia. They dislike going out of doors in the sunlight, and are intolerant to bright lights. In such situations they "squint", or close the eyes altogether. Light usually provokes excessive lacrimation. Or, they may be brought to the pediatrician because of recurrent, mild episodes of "red eyes." There may be little objective evidence other than conjunctival or episcleral injection, usually circumcorneal injection, and non-purulent lacrimation. This stage is often indistinguishable, clinically, from a mild allergic conjunctivitis. If it progresses, corneal changes supervene, as described above.

In the third group, there is antecedent ocular pathology, such as hemorrhage into the anterior or posterior chamber, virulent infection, retrolental fibroplasia, aniridia, tumor, etc. These cases are rarely seen by the pediatrician, since the antecedent pathology usually places the child under the care of the ophthalmologist. The one exception is anterior chamber hemorrhage following trauma. The management of this type is not within the scope of the pediatrician.

DIAGNOSIS

The first step in the diagnosis of glaucoma is an awareness or consciousness of the glaucoma problem. The eyes should be examined as carefully as other systems during every routine examination; and consideration should be given to the mother's observations concerning the child's eyes and vision. Errors of omission are made because the mother's observations are dismissed as being due to "overanxiety." A high index of suspicion is warranted in cases presenting unusual photophobia and lacrimation, or recurrent episodes of ocular, non-purulent, inflammation. Of course, once anatomic abnormalities have occurred, diagnosis depends upon pure observation.

Congenital glaucoma is unilateral in 33 per cent of cases. Thus in two-thirds of these children complete blindness is a distinct possibility. Since

treatment, if to be effective, must be started early, prompt diagnosis is imperative.

The absolute diagnosis of congenital glaucoma rests on two procedures. First, instrumental tonometry, done under general anesthesia, provides the *sine qua non* of diagnosis, and allows the ophthalmologist to follow the effect of treatment. Second, gonioscopy, that is, examination of the chamber angle and trabecular meshwork by means of a prism, confirms the diagnosis and provides the means of visualizing the angle during corrective surgery. Measuring the corneal diameters should be mentioned as an ancillary procedure, although it is recognized that glaucoma may be present even though the corneal diameters are normal. The average corneal diameter of the newborn is 9.5 mm.

Glaucoma may be confused with few ocular diseases. In megalocornea the eye looks large due to the increase in size of the cornea. However, the anterior chamber is shallow and the tension is normal. Probably the most difficult clinical differentiation to be made is in an allergic conjunctivitis. Here there is photophobia and excessive lacrimation; the tension is normal however. Also, recurrent serous conjunctivitis may resemble early glaucoma. The tension and gonioscopy examinations, and response to antimicrobial or antiviral treatment establishes the diagnosis. Any questionable case should be submitted to tonometry.

TREATMENT

1. *Infantile Glaucoma:* All cases exhibiting congenital anomalies of the chamber angle should have surgery as soon as possible after the diagnosis is made. The operative procedure has as its purpose the opening up of the filtration angle so the aqueous has access to Schlemm's Canal. The importance of early surgery can be appreciated from Table I which compares the residual vision between the operated and the non-operated group. As a matter of practice, the ophthalmologist will do a goniotomy at the same time that the tension is taken. Within a week of the goniotomy, the infant is anesthetized again, the tension recorded and, if still elevated, reoperated. Often, several procedures are needed to normalize the tension. It should

TABLE I

	Group Operated During First Year of Disease	Unoperated Group
	%	%
Cases with 20/40 vision after 2 yrs.	Less than 10	
Blind by 12 years	25	54
Blind by 25 years	45	60

be evident to the pediatrician that the age of the infant is not a factor to be considered in deciding for or against surgery. The younger an infant with glaucoma is operated, the better the chances for preservation of vision. Infants with congenital glaucoma should not be given drugs of the atropine-belladonna group. These drugs may produce mydriasis, which, by folding the iris into the chamber angle, may completely obstruct the egress of aqueous, thus causing an acute congestive type of glaucoma, sending the tension to maximal heights.[3] As mentioned above, the glaucoma associated with angiomatosis may be due to hypersecretion of aqueous. In these cases, if the chamber angle is normal, the formation of aqueous may be inhibited by means of acetazoleamide, (Diamox).[4] Obviously, the glaucoma associated with retrolental fibroplasia is incurable except by enucleation. This may be done for corneal ulceration with infection or severe pain.

2. *Juvenile Glaucoma:* The treatment of this group depends entirely on the cause of the glaucoma. Mietrics, mydriatics, and acetazoleamide (Diamox) may be used, for specific indications.

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LEAD POISONING IN CHILDREN

A Review of 17 Cases at the Children's Hospital, Washington, D. C.

Thomas J. Ryan*

Although lead poisoning is a well known hazard in many industries, rarely is its significance as a serious pediatric problem fully appreciated. In this group it has as appreciable incidence and thus constitutes one of the greatest poisoning problems in children. A survey of the case records at Children's Hospital, Washington, D. C. reveals, that from 1950 to 1954 there were seventeen cases of plumbism admitted to the hospital with one fatality. In view of the important differential diagnosis associated with this condition, the comparative ease of establishing the diagnosis on history alone and the success of present day therapy, it seems worthy to review the subject and to stress the importance of having the entity in mind when confronted with the acutely ill, convulsing or severely anemic patient.

INCIDENCE

Since lead poisoning is not a reportable disease, there is a lack of adequate morbidity data. However, the condition is found widespread throughout

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TABLE I

Age Incidence

Hospital	Age	Cases
Children's Memorial—Chicago	18 mos.–4½ yrs	22
Johns Hopkins—Baltimore	13 mos.–7 yrs	22
Boston Children's	12 mos.–5 yrs	89
Hahnemann Hospital—Philadelphia	16 mos.–4 yrs	12
Children's Hospital—Washington	16 mos.–4 yrs	17

the United States, naturally being reported more frequently from the larger cities, especially, Boston, Baltimore and Cincinnati. There were 220 cases reported by Williams in Baltimore during the past eighteen years with 18 deaths. Kotte of Cincinnati, reported 59 cases from 1928 to 1950 in that city. In Philadelphia there were recently reported 12 cases occurring in a period of 22 months, with four deaths. At the Children's Hospital, Washington, D. C., there have been 17 cases in the past four years. Plumbism has its highest incidence in children between the ages of 1½ and 3½ years and corresponds to that age group where objects are put in the mouth almost as a matter of routine. Of the 17 cases reviewed here, the youngest child was 16 months, the oldest, 4 years, with a mean age of 2 years. Table I lists the age incidence reported by various hospitals throughout the country.

There is no significant racial or sex predominance, although lead poisoning will be found more commonly among the lower economic classes. It has been pointed out that there is a seasonal incidence in the warm months from June to October, with an increased incidence of encephalopathy during the Spring and Summer months. Rappoport relates this to the intake of vitamin D and sunlight and concludes that a vitamin D producing agent, either sunlight or cod liver oil, increases the amount of lead absorbed from the gastrointestinal tract. The photosensitization of sunlight on porphyrins, which are present in increased amounts in patients with plumbism, may play a role in contributing to the severity of the disease. Of the 17 cases reported here, 15 occurred between June and October and all but two of these gave definite manifestations of encephalopathy, that is, marked irritability and/or generalized convulsions.

SOURCES OF LEAD

The most common source of lead intoxication in children is lead-containing paint. Although today, lead-containing paint is no longer used on children's furniture, toys and all indoor woodwork, there is considerable contact with hand-me-down, repainted toys, reused nursery furniture and repainted indoor woodwork, all containing several coats of lead. Fre-

quently there is a history of the infant peeling the wallpaper by his crib that has been painted over with lead paint or chips of plaster in the same situation. Indeed, one infant in our series had a favorite "hole in the wall" that he chewed to a size of 3′ x 1′. Lead toy soldiers also represent a common source. Among the less common sources are lead nipple shields, popular a decade ago, but since banned, and, in Japan, what was first thought to be a "new type of meningitis" was shown by subsequent studies to be the result of a lead-containing body powder that mothers were using on their breasts. The inhalation of lead vapors sufficient to cause intoxication in children has occurred frequently from the burning of lead battery casings for fuel in the home. There was a considerable outbreak of this in Baltimore in 1932.

PATHOLOGICAL PHYSIOLOGY

It is the balance between the absorption, storage and excretion of lead that determines its toxicity. The most common route of absorption in children is by way of the gastrointestinal tract, being explained by the phenomenon of pica, and absorption here depends more on the length of time that the lead is in the intestine, than on the dose ingested. Thus a dose of 30 mgm. may well be innocuous as a single dose, but 0.3 mgm., taken daily, over a period of time, is certain to produce intoxication. The small amount of lead salt that is soluble in the intestine is absorbed in the upper duodenum and enters the portal circulation; the remaining quantity is not absorbed and is excreted in the feces. The lead in the portal circulation passes to the liver where the majority of it is excreted in the bile. The remainder enters the systemic circulation and is deposited either in the bones, as an insoluble tertiary phosphate along with calcium, or in the soft tissues, especially the liver, kidney, pancreas and brain. The chief clinical problem is lead deposition in the brain tissue where it causes an encephalitis with acute cerebral edema, widespread vascular damage, disturbance of brain cells, particularly in the cortex and cerebellum, and neurologic disintegration. The occurrence of symptoms depends upon the amount of lead in the tissues and in transport from bowel or soft tissue to bone, or from bone to soft tissue. It is fundamental to understand that mobilization of lead from tissues to bone parallels calcium deposition in bones and that decalcification is associated with the release of lead from the bones. Thus conditions such as diet, infection and acidosis will exert a profound effect

TABLE II

Blood and Urine Levels

Urine	.02–.08 mgm./L
Blood	.4–.06 mgm./100 ml

upon the circulating blood level of lead. Once the source of lead is removed, the lead in the bones and tissues gradually is excreted from the body by normal physiological processes. The amount of lead in the urine roughly parallels the amount of lead in the blood and thus serves as in indication of the rate of transport. Table II gives the normal blood and urine values.

SIGNS AND SYMPTOMS

a) *Acute Lead Poisoning:* This is a rare entity in children and is caused by a sudden, overwhelming exposure to lead, such as in the inhalation of fumes from burning of storage battery casings. It is characterized by an abrupt onset of symptoms with nausea, vomiting and abdominal pain. There may rapidly occur muscular weakness, pain or paralysis. Hemolytic crisis can occur and death in such cases is not uncommon.

b) *Chronic Lead Poisoning:* Because children are more inclined to ingest small amounts of lead over a long period of time, chronic lead poisoning is the picture most frequently seen and, indeed, is the only type seen in the 17 cases analyzed here. As stated above, the symptomatology of lead poisoning is dependent upon the physiological transport of the ingested lead from bowel or soft tissue to bone and, vice versa. Not only are the symptoms therefore intermittent, with frequent exacerbations, but they are directly influenced by any metabolic factor that mobilizes the lead that has been previously deposited in the bones and tissues. The history in most instances is that of periods of well being interrupted by periods suggestive of poisoning.

As with most disease states, the single factor most important to making the correct diagnosis is proper history taking. More emphatically, because it is a problem of intoxication, the history of ingestion or exposure to fumes becomes a *sine qua non* for diagnosis. This history can be considered substantiated by the mere confirmation of pica in the child, so widespread is the source. This is illustrated beautifully in 4 of our 17 patients where the history of pica alone was the motivating cause for further workup. All four were proven to have lead intoxication by x-ray, blood picture and response to therapy.

Chief among the symptoms of chronic lead poisoning are irritability, convulsions, pallor, anorexia and vomiting, usually with an insidious onset. The most prominent and alarming symptoms are those referable to the central nervous system and, while they do not appear until the disease has existed for some time, they may appear suddenly, in association with some secondary disturbance producing a rapid release of large amounts of lead. The convulsions are usually generalized, repeated and difficult to control. The one fatal case in this report had status epilepticus for twelve

hours prior to admission and after vacillating between coma and convulsions for four days in the hospital, she expired, in spite of the most vigorous therapy. Involvement of the central nervous system is manifested not only by irritability and convulsions but also by disorientation, hemiparesis, general flaccidity, hyper-reflexia, gross tremors and pathological reflexes such as Kernig's and Babinski's.

The gastrointestinal tract is almost invariably involved, producing the conditions of anorexia, vomiting, diarrhea and abdominal pain frequently accompanied by abdominal distension. Most authors allude to the frequency with which constipation is a presenting symptom. It is interesting to note that in our 17 children, not one complained of constipation but three came with a chief complaint of diarrhea.

Two additional symptoms seen in a significant number of our 17 patients, not frequently mentioned in the literature, are worthy of comment. Leg pain and swelling of the hands, feet or face were found in seven of the cases. The leg pain was not specific, occurring in the muscles as well as in the joints. The swelling of the hands, feet and face was described as soft tissue swelling and periorbital edema.

Physical examination in general is not contributory, other than the manifestations of central nervous system involvement, already described above. The body temperature can be normal or increased. Anemia, while invariably present, seems to escape detection by the present day clinician and was noted usually only on the laboratory sheets of the charts reviewed. This leads one to question the frequently made statement that the pallor seen in lead poisoning is out of proportion to the degree of anemia. Tachycardia was usually seen and is explained by the anemia present. It is generally agreed that the lead line, popular because of its description in the text books, occurs in less than 2 per cent of the cases of lead poisoning in children and is never present in infants without teeth. In not one of the 17 children was the lead line described, most of the charts explicitly stating so in the negative. Table III is a composite of the symptoms and physical findings seen in 17 patients. The symptoms are counted on the number of times that they appeared in the chief complaint.

TABLE III

Signs and Symptoms

Irritability	7 cases	Leg pain	3 cases
Convulsions	7 cases	Swelling of hands, feet or face	4 cases
Drowsiness	5 cases	Hyper-reflexia	6 cases
Disorientation	5 cases	Gross Tremors	2 cases
Anorexia	7 cases	Paralyses	2 cases
Vomiting	6 cases	Hyperthermia (103 plus)	4 cases
Abdominal pain	4 cases	Abdominal distention	2 cases
Diarrhea	3 cases		

TABLE IV

Basophilic Stippling

	Cases	%
Children's Memorial Hospital, Chicago	22	81
Johns Hopkins	22	100
Hahnemann Hospital	8	66
Children's Hospital	9	53

LABORATORY DATA

The most positive laboratory evidence of lead poisoning consists in the demonstration of excess amounts of lead in the blood and urine. This is a difficult and time consuming task. Additional reliable laboratory data can be obtained more easily.

1. *Blood:* Anemia is invariably present, of the hypochromic variety. It is believed to be due to the actual destruction of the cells by lead. The hemoglobin values in our 17 patients ranged from 6.8 grams to 12.8 grams with a mean value of 9.5 grams. The hematocrit values ranged from 25 per cent to 38 per cent with a mean of 30 per cent. Basophilic stippling, while a non-specific finding, aids considerably in arriving at the diagnosis when present along with other findings. One of our cases was picked up in a child who was admitted because of otitis media and who showed stippling on the smear of a routine blood workup. It is not by any means an invariable finding, being seen in only 9 of our 17 children or in 53 per cent. Table IV shows the percentage of children showing stippling in four different series.

2. *X-ray*: The x-ray findings in chronic plumbism are almost a constant, although late, finding except when active rickets is present. Roentgenograms of growing bones reveal deposits of tertiary lead phosphates as heavy transverse lines in the ends of the shafts of long bones. These metaphyseal lines are similar to growth lines except that the lead lines are heavier and thicker.

3. *Urine:* The porphyrin test in the urine will frequently be positive in lead poisoning. A specimen of urine examined by means of an ultraviolet light will give off a fluorescence.

COURSE AND PROGNOSIS

Spontaneous recovery is possible if the patient is removed early from the source of the lead, but, if lead ingestion continues, there will be marked involvement of the brain with death or at least considerable residual, which manifests itself especially in the form of behavior disturbances, mental deficiency and recurring convulsions. Under present day therapy, the duration of hospitalization tends to grow shorter. Table V presents the duration of hospital stay of 17 cases under therapy.

TABLE V

R. D.	10 days	A. J.	11 days
D. H.	5 days	B. B.	mort
D. J.	19 days	E. M.	14 days
J. D.	20 days	S. S.	2 days
C. C.	8 days	W. R.	23 days
A. J.	10 days	J. S.	2 days
F. G.	18 days	M. J.	16 days
S. S.	15 days	L. E.	5 days
		R. P.	13 days

TREATMENT

The present day therapy of lead poisoning results from the recent discovery of several substances that mobilize lead from the tissues and transport it in the form of a non toxic, non-ionizable complex, to the kidney where it is excreted. Thus the earlier regimens of first inducing the deposition of lead in the bones and tissues to reduce the toxic blood levels and then instituting carefully controlled deleading processes once the acute phase has terminated, are no longer necessary.

BAL, 2-3 dimercaptopropanol, has been employed by Ennis and Harrison on the grounds that this drug has shown great value in the treatment of other heavy metal poisonings, such as mercury, arsenic and gold. They recommend the dosage of 2–4 mgm./kg given for a period of 10 days, using a 10 per cent solution in benzyl benzoate and oil. Belknap, however, feels the lead-BAL complex is not as stable as the other heavy metal complexes and is of little help in the treatment of lead poisoning.

In Philadelphia, sodium citrate is still the treatment of choice after the fashion of Key and Letonoff. Citrate forms a soluble complex of extremely low dissociation with lead and after the oral ingestion of 1–2 gms., three times a day, there is noted a rapid subsidence of acute symptoms, with a moderate decrease in the concentration of lead in the blood and an increase in the urine. This treatment should be maintained for periods upward of two months.

At this hospital, Bessman, Rubin et al., have employed EDTA (ethylenediamine-tetra-acetate) with such profound success that it has become the drug of choice. The calcium salt, calcium disodium versenate, has been given subcutaneously in the dosage of 30 mgm. every eight hours for five days and, after a 2–3 day rest, repeated. However, the final dosage and schedule of administration of this drug, awaits further publication by these workers.

In addition to these specific methods of treatment, there remain of course, the usual supportive measures. Sedation, analeptic medication, blood transfusions and even cranial decompression are frequently indicated. Convulsions can be managed by magnesium sulfate, .2–.4 ml. of a 25 per

cent solution per kilogram of body weight given intramuscularly every 6 hours will often control this problem before there is a dangerous fall in blood pressure.

SUMMARY

1. Lead poisoning is still an important problem in pediatrics which can be accompanied by fatal and irreversible sequellae and not infrequently occurs in unrecognized and untreated cases.

2. Lead containing paint on furniture, toys, woodwork and other household objects, is the main source of this toxic condition.

3. The chief clinical problem of lead poisoning is lead deposition in the brain tissue and the occurrence of symptoms depends on the amount of lead in the tissues and in transport from the site of absorption in the upper duodenum.

4. The most essential requisite for making the diagnosis of plumbism is the history of pica. The symptoms are insidious in onset and include, chiefly, irritability, convulsions, pallor, anorexia and vomiting.

5. In addition to finding elevated lead levels in blood and urine, other findings in the blood and urine are discussed. The x-ray picture of lead lines in the metaphyses are considered diagnostic.

6. The overall prognosis is good, but fatalities are not uncommon and there is an appreciable incidence of serious sequellae.

7. Treatment consists of specific and supportive measures. Among the former, BAL, sodium citrate and calcium disodium versenate, are discussed.

8. This paper was based on a survey of the records of 17 patients with lead intoxidation at Children's Hospital, Washington, D. C. The recent literature is reviewed.

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MENINGITIS DUE TO CONGENITAL DERMAL SINUS

R. L. Bregman, M.D.

CASE REPORT

S.M.* a 4 year old white male, was in apparent good health until six to eight weeks prior to admission, when he developed an upper respiratory infection which responded to one injection of penicillin. Four days before admission he vomited forcefully, became febrile and he also developed a stiff neck and pain in the legs.

The past medical history was essentially negative, except for frequent complaints of leg and foot pain and vague rectal pain.

Physical examination on admission revealed a cooperative white male with a temperature of 102°, who did not appear severely ill. There was red pigmentation of the skin in the lower midline of the lumbar region posteriorly overlying the vertebral column, measuring 2 cm. in diameter. In the center there appeared to be a sinus tract. The area dimpled when the skin was pulled and pouched-out when pressure was exerted. There was no local drainage and no apparent infection.

Neurological examination revealed back and neck rigidity and a positive Kernig sign. Deep tendon reflexes were hyperactive, sensory changes were not evaluated. There were no other physical abnormalities.

Lumbar puncture revealed cloudy fluid with 2,100 leukocytes, 90 per cent polymorphonuclears and 10 per cent lymphocytes. A gram positive diplococcus was cultured from the spinal fluid. However, the organism could not be grown on subculture.

The patient was treated with aureomycin and chlormycetin intravenously and penicillin intramuscularly. He did well until the fifth hospital day when his temperature rose to 103.5°, and the spinal fluid was found to contain 290 leukocytes with 64 per cent polymorphonuclears and 36 per cent lymphocytes. Fever continued, with several spikes to 105°. Physical examination was within normal limits. Blood cultures were negative. The only significant finding was the dermal sinus. Lumbar puncture was repeated on the eighth hospital day revealing 1,890 cells with 56 per cent polymorphonuclears and 44 per cent lymphocytes. X-ray examination of the lumbar spine revealed a spina bifida occulta of the fifth lumbar vertebrae which was thought to be of no pathologic significance.

On the ninth hospital day a laminectomy was performed on L3 and L4. The dermal sinus was excised, the sinus tract was traced into the dura where a cystic mass was found which extended from L3 to S1. The pathologist reported a dermal sinus and connected cyst.

The patient became afebrile on the fourth postoperative day and made an uneventful recovery. Antibiotic therapy was continued for one week postoperatively and no further lumbar punctures were done. No neurological abnormalities were noted at the time of discharge.

Eighteen days after discharge from the hospital the child was given an injection of gamma globulin after being exposed to measles. He had been asymptomatic and was doing very well according to his parents. Following the injection of gamma globulin he vomited several times, had chills and fever and complained of stiffness in the neck and legs.

On admission the temperature was 102° but he was alert and comfortable. Neurological examination revealed minimal nuchal rigidity and spasticity of the back muscles. The deep tendon reflexes were most active on the right side and there was a positive Brudzinski sign. There were no abnormal sensory changes.

* Patient from the service of Dr. Fredric G. Burke.

Lumbar puncture in the region of L4 revealed 1,800 leukocytes with 90 per cent polymorphonuclears. There was no evidence of block and the spinal fluid pressure was within normal limits. No organism could be grown out of either the blood or spinal fluid cultures. Treatment consisted of intravenous chlormycetin (after a penicillin reaction) following which the patient became afebrile in 48 hours. Repeat lumbar puncture one week after admission revealed 77 cells with 85 per cent lymphocytes. The spinal fluid obtained on the eleventh hospital day contained 42 leukocytes with 94 per cent lymphocytes.

The patient was discharged without any evidence of neurological disease. The second episode was thought to be a reactivation of the original infection.

This subject is being reviewed as a result of a case of meningitis associated with a congenital dermal sinus in the lumbar region, recently seen in the Children's Hospital. That this condition is relatively rare is borne out by the fact that there are only approximately fifty cases reported in the literature. It is probable that the condition is commoner than published reports would indicate. Often, the more rare conditions are not considered when one is making a diagnosis. This factor becomes important in cases of congenital dermal sinuses, since, if the diagnosis is made early, before complications occur, the treatment is simple and the prognosis is excellent.

Congenital dermal sinuses are dermal tubes, lined with stratified squamous epithelium, which extend from the skin surface for varying distances and frequently extend to the central nervous system or its coverings.

The inner end of the sinus may expand to form a dermoid or epidermoid cyst. The epidermoid cyst contains only epithelial debris and is lined with stratified squamous epithelium, whereas the dermoid cyst contains sebaceous material and hair and the lining includes elements of deeper layers of skin. Histologically this resembles the common pilonidal cyst, which has no communications with the spinal cord and meninges. It differs from the pilonidal sinus in that it may occur anywhere between occipital and sacrococcygeal region and in its connection with the central nervous system or meninges.

Congenital dermal sinuses result from failure of proper separation of the neuroectoderm from the epithelial ectoderm which normally occurs between the third and fifth weeks of intrauterine life. This process normally is initiated along the mid-dorsal aspect of the embryo and the anomalies may occur from the occiput to the coccyx. These sinuses are more common in the lumbo-sacral region; those in the occipital region may be easily missed if the scalp is not shaved.

The majority of cases occur in children under the age of five years; however, the first case, reported in 1926 by Moise, was an 18 year old with meningitis and a discharging sacral sinus.

Congenital dermal sinuses are significant, primarily, because of the complications they produce. Bacteria may enter the central nervous system via the congenital dermal sinus at any point from the occipital region to

the coccygeal region. As a result, meningitis, subdural abscess, intramedullary spinal cord abscess, or osteomyelitis may occur. Subcutaneous, paraspinal, or epidural abscesses may also occur.

CONGENITAL DERMAL SINUS IN THE OCCIPITAL REGION

Congenital dermal sinuses in the occipital region are characterized by a small dimple or sinus opening in or near the midline of the scalp. Hair may be absent for 1–2 cm. around the sinus opening and a port wine stain of the surrounding skin may exist. The scalp may be thickened and a small, palpable, movable, subcutaneous mass may be present.

In addition to the presence of the sinus, pressure symptoms may arise from the cystic mass formed by the sinus, or it may become infected. The clinical picture, therefore, may present itself as a cerebellar or fourth ventricle tumor, obstruction hydrocephalus, or meningitis. The patient may have headache, vomiting, ataxia, nystagmus, papilledemas, extraocular motor weakness, enlargement of the head or acute meningitic symptoms. *Staphylococcus aureus* is the most common organism associated with meningitis secondary to congenital dermal sinus in the occipital region; however, in many cases no organism is found.

According to Matson and Ingraham a defect in the bone always exists in such cases. This may or may not be demonstrated by roentgenograms. They state that any child with a midline dimple or mass in the scalp should have skull x-rays. In addition, any child with unexplained meningitis, especially if due to *Staphylococcus aureus*, should have the entire midline of the scalp as well as the skin of the back examined for sinus openings. These authors further state that in the presence of a midline occipital dimple, increased intracranial pressure probably is indicative of cystic expansion of the sinus with resulting obstruction within the posterior fossa. Surgical exploration is indicated in such a case.

CONGENITAL DERMAL SINUS IN THE LUMBAR REGION

Patients with a congenital dermal sinus in the lumbar region often give a history of redness, swelling and tenderness in this area. They may also have a thin purulent discharge from the sinus opening. Physical examination may reveal local findings similar to those described for the occipital lesion. The sinus may not be detected until meningitis occurs, or in some patients, until there have been recurrent attacks of meningitis, and the anomaly is specifically looked for.

The most common organism in this type of congenital dermal sinus is *E. Coli* although *Staphylococcus aureus* and *Streptococcus Fecalis* also occur.

The signs and symptoms may be those of any meningitis or may be due to an intraspinal expanding mass with weakness, paresthesia, pain, sphinctor disturbance and sensory or reflex disturbances.

Roentgen examination usually reveals a spina bifida or grooving of the upper or lower surface of a single spinous process; however, at times the vertebrae may be normal. This defect may be more difficult to see in infants.

TREATMENT

The treatment of congenital dermal sinuses, regardless of location, is complete excision of the entire dermal sinus tract from the skin surface to its deepest projection, including all cystic expansions, before infection occurs.

In the presence of local skin infection or meningitis without neurological abnormality, operation should be deferred until the infection has been treated adequately with antibiotics and local therapy given. If intraspinal extension is suspected, it is unnecessary and unwise to probe the sinus tract or inject with any type of tracer dye.

Bucy in discussing Mount's paper stated that the presence of a dimple in the midline of the back overlying the spine is indication for its removal. He also points out that pediatricians often noted this anomaly but passed it off as an unimportant finding.

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ABDOMINAL AUSCULTATION IN THE NEWBORN AND INFANT

John H. Bayly, M.D.

I have observed that few physicians auscultate the abdomen. This omission is not limited to pediatricians, it is also omitted by surgeons. The reason for this negligence begins in medical school. A professor of medicine recently told me that auscultation of the abdomen "probably has very little value." Medical students and internes are not duly impressed with the value of auscultation as they seldom see it practiced. Medical students have the same trouble in identifying peristaltic sounds as they do with pulmonary sounds. All of these sounds must be heard to be appreciated, inasmuch as complete verbal description of sound is difficult. Much emphasis is laid on an x-ray examination of the chest when doubtful findings are present. This may make one wary of his ability or shortcomings in auscultation of the chest. Many observers order x-rays of the abdomen rather than relying on physical examination. I am certain that the physician should

not throw away his stethoscope because x-ray is available. The stethoscope is an important adjunct in abdominal examination. An early central pneumonia may better be noted more easily by x-ray than by auscultation. An early obstruction may be diagnosed by auscultation 8–12 hours before the x-ray findings may be positive. In some cases roentgen study may never become positive. The classical case of appendicitis may not need confirmation by auscultation. Routine auscultation in a suspect appendicitis may reveal a Richter's hernia or other pathology and prevent the face saving statement, "well the patient needed the operation although the diagnosis was wrong."

Abdominal auscultation can improve diagnostic accuracy and make earlier diagnoses. Auscultatory findings may permit greater latitude in time for observation of abdominal complaints. Auscultation may suggest medical problems such as diarrhea, colic, and constipation. Surgical complications may be diagnosed sooner. The progress of a post operative patient with indications for gastric suction, enemas and feeding may be assayed by means of the stethoscope. It may be the most important method for final diagnosis.

Auscultation in physical examination of the abdomen may be done first. In newborns and infants to avoid rousing them the use of a warm stethoscope is advised and the practice of auscultation should be carried out before palpation and percussion.

Auscultation of the stomach is usually practiced more satisfactorily with the patient in a sitting position. The newborn or infant will usually stop crying when given the bottle. Fluid normally has a "slithering" sound as it enters the stomach. In gastric distention the sound of fluid entering the stomach is likened to the dropping of a stone into a deep well. A loud "poong" of high resonance is produced.

Gastric distention due to ingestion of air is an important problem to the pediatrician. This excess air may cause the loss of the feeding when the baby is "burped." It may result in vomiting which can be projectile. It may give premature satisfaction of feeding to the infant. In cyclic vomiting associated with a dilated stomach, several feedings may be retained before vomiting occurs.

Surgeons are concerned because of the acute shock which gastric distention may create—such shock may be sufficient to cause a death. Tracheobronchial aspiration frequently occurs with resultant severe vomiting. One may be lulled into a false sense of security when a post-operative infant takes several feedings and then appears to do poorly without apparent reason. Vomiting or gastric suction may quickly improve his picture. Auscultation will indicate the adequacy of gastric suction. Gastric drainage tubes require constant observation. Ausculation is more simple and cheaper than x-ray studies and avoids transportation and handling of a sick baby.

Normally, small intestinal peristalsis is best heard about 3.0 cm. below the umbilicus. Large bowel sounds are best noted along the distribution of the colon. Audible peristalsis in the newborn varies in the time of onset. The earliest I have heard such sounds was at seven hours. In some newborns 12 to 24 hours may elapse before it is heard. A vigorous healthy baby that is born after a minimum of anesthesia and sedation to the mother is apt to swallow air earlier than a weaker child born with more drug effect. The frequency of sounds will depend on activity of the intestine at the time of examination. Factors that speed up peristalsis include recent ingestion of food, drinking cold fluids, and catharsis. Inhibiting factors include starvation, vomiting, and the effects of toxemia.

Normal sounds are usually heard immediately and 10 to 15 seconds in a routine examination may be all that is necessary. It is unusual to wait more than 2 to 3 minutes to hear normal peristalsis. Extended periods of time may be necessary to note abnormal peristalsis, and this is a good reason to make auscultation at the beginning of an abdominal examination. As one inspects, palpates and percusses, the stethoscope may rest on the abdomen until identifying sounds are heard. In selected cases, the giving of water or formula may stimulate peristalsis. Abnormal peristalsis in the upper small bowel may not be noted for intervals up to about 5 minutes and in the terminal small bowel may require up to 20 minutes.

Peristaltic sounds heard include hissing, bubbling, squealing, sounds like blowing air through puckered lips at low pressure. They may vary in pitch. These sounds often appear to be disconnected and without pattern. It is important to know when these sounds become abnormal. They are easier to appreciate and describe as one practices listening to them.

We classify abdomens that are quiet, silent, noisy, or obstructive. A quiet abdomen is usually found in one who has not eaten in the preceding 6 hours or recently has vomited. In this situation one hears nondescript faint sounds. They are easily differentiated from the sounds of the transmitted pulse, heart, lungs or aneurysms. They are often so faint that description is impossible. No peristalsis may be noted in severe forms of gastro-enteritis without vomiting or diarrhea. No peristalsis may be noted with irritation of minimal amounts of blood or cystic fluid in the peritoneal cavity which may cause paresis of the intestine. The quiet abdomen must be observed closely to note changes. In functionally quiet peristalsis atropine may relax the intestine and permit peristalsis to recur. A quiet abdomen may be the harbinger of developing disease.

We may consider now the changes which take place in appendicitis and resulting abscess. This will demonstrate the slow progressive changes typical in inflammatory diseases of the abdomen. Appendicitis in all age groups may produce only mild symptoms and signs. It is the progression of these symptoms and signs that establishes the diagnosis. Auscultations repeated at

intervals usually gives characteristic findings and indicates the necessity for operation or change in diagnosis.

Unlike the adult, the infant does not have a thick, well developed omentum to wall off an infectious process. The onset of the disease in many cases may be associated with diarrhea. Infants have a more active intestinal tract than do adults. Incomplete rotation may place the appendix in an unusual area.

In the usual appendicitis, where the appendix is free in the peritoneal cavity the following train of events may be followed when an abscess does develop. At early onset peristalsis may be hyperactive. While good active peristalsis is present one can safely continue observation of the infant while waiting to clarify the diagnosis. This is particularly important when one suspects the pain may be due to conditions such as worms, constipation, diarrhea, or pharyngitis.

As inflammation progresses the abdomen usually becomes more quiet. Faint nondescript sounds may be heard which are not diagnostic. This is a period of increasing suspicion, but no alarm or hurry is necessary. This represents the so-called "quiet abdomen."

As infection penetrates the appendiceal wall, the serosa of the appendix is inflamed. This irritant to surrounding intestine causes the bowel and omentum to wall off the appendix. A localized ileus occurs. The abdomen remains quiet. The bowel loops involved in the walling off process become dilated and atonic. The remainder of the small bowel is potentially active. The taking of food or fluid at this stage may encourage peristalsis. The administration of cathartics at this stage would whip up peristalsis and break down a protective barrier.

Auscultation now discloses isolated tinkles in an area about the appendix with an otherwise quiet abdomen. These tinkling sounds are produced by mild peristalsis which empties intestinal contents from above into a distended loop of bowel containing fluid and gas. This isolated ileus with its tinkles now gives final support to the diagnosis of appendicitis.

As the disease progresses to abscess formation the sounds alter. Because of the loop of paralyzed bowel, peristalsis becomes loud, is heard in distinct rushes, and is associated with mild to moderate crampy pain. These rushes sound like an ocean wave with irregularity in time. High pitched tinkling sounds persist at the end of the wave. This is not to be confused with the high resonant sounds of an aerophagic. The intestinal contents are passively pushed thru an antonic loop of bowel by proximal hyperactive peristalsis. A flat x-ray film of the abdomen at this stage would give the appearance of an incomplete obstruction. Distention of the patient at this stage may be moderate and vomiting may be absent. The auscultatory signs would be similar to that found in early volvulus of a segment of small bowel where

vascular impairment to the loop causes ileus. Drainage of an abscess will usually relieve the obstruction.

Because of the absence of early signs, mechanical obstruction of the bowel in the newborn may not be suspected until 48 to 72 hours after birth. Auscultation may demonstrate the diagnosis 8 to 12 hours earlier than x-ray in most instances. Earlier diagnosis may be made in acute mechanical obstruction of infants too.

The peristaltic sounds depend on the location of the obstruction, the degree of obstruction and elapsed time of obstruction. Newborns with obstruction of the lower ileum continue to swallow air although they may not retain water or the feeding. Obstructive peristalsis may not occur for 24 to 36 hours. The sounds occur in rushes like the ocean waves breaking on the shore. Between rushes the abdomen is quiet. With each rush the infant will show evidence of pain and then relax as the evidence of peristalsis disappears. This triad of a quiet abdomen, rush of peristalsis, and apparent pain is diagnostic of small bowel obstruction. Gastric suction at this stage will eliminate the peristaltic rushes and produce a silent abdomen.

The peristaltic rushes become less active as distention progresses from above the point of obstruction and the bowel wall is stretched. Tinkles, high pitched and metallic, persist in advanced obstruction. These sounds are elicited when abdominal pressure is exerted, thus forcing fluid from one loop to another. Turning the patient from side to side will produce the same sounds.

In jejunal obstruction distention is not marked. The fluid and gas is easily emptied into the stomach where it may be auscultated unless vomiting has emptied the organ. A faint gurgle of peristalsis associated with apparent pain by the baby is noted. This occurs in an otherwise quiet abdomen. In incomplete obstructions due to Richter's type of hernia with small incarcerations at the internal ring we hear obstructive peristalsis. Usually rushes of peristalsis and pain occur and because the lumen is not completely obstructed, air and fluid continue to pass and the abdomen is not quiet. Here again x-ray findings may show only moderate gaseous distention that would only be suspicious. A volvulus of the sigmoid colon may well be similar. A congenital band may completely obstruct the lumen giving characteristic auscultatory sounds as described. When gangrene ensues peritoneal irritation caused by the transudate and infection produces a silent abdomen which with ingestion of air gives high pitched tinkles on passive movement of the intestines.

A silent abdomen gives no peristaltic sounds. This is usually due to a severe chemical irritant such as follows a ruptured peptic ulcer, perforated intestine which may occur in meconium ileus, ruptured appendix with peritonitis, or massive peritoneal hemorrhage. A silent abdomen present

in the newborn which persists after 12 to 24 hours should be regarded as pathological.

Constipation is the most common cause of abdominal pain in a child's abdomen. Auscultation in these cases reveal a constant "ticking" sound that is almost pathognomic when found. Although constipation is frequently present at the onset of appendicitis, I have never heard the "ticking" sounds in any case of appendicitis. An enema given within about three hours will produce tinkles, splashes and weird sounds that are confusing.

Colic in a baby is recognized by the constant explosive resonant sounds that are heard.

Gastro-enteritis usually gives the noisiest abdominal sounds. Even though diarrhea may be present with the onset of appendicitis, peristalsis is not usually violent. With gastro-enteritis pain decreases as peristalsis quiets down under treatment. With appendicitis the pain is increased as peristalsis subsides or an ileus develops.

The differential diagnosis between mesenteric adenitis and appendicitis is difficult. These patients may be treated by parenteral fluids and antibiotics and observation continued when active normal peristalsis continues.

Intestinal parasites commonly produce abdominal pain associated with active peristalsis. Worms may also produce obstruction with characteristic auscultatory signs. Abdominal pain seen in acidosis associated with vomiting often produces quiet peristalsis. While correcting the loss of fluid intake the vomiting disappears, anorexia disappears and active peristalsis recurs as the pain is controlled.

Respiratory infections in children are sometimes associated with lower abdominal pain. The peristalsis is usually active.

Other diseases may give abdominal pain suggesting intra-abdominal inflammation. These include Henoch's purpura, sickle cell anemia and infectious lymphocytosis. Active peristalsis is present.

Wilms' tumor may present itself with abdominal pain due to retroperitoneal hemorrhage. Active peristalsis permits study of the patient rather than apprehension concerning an intra-abdominal catastrophe.

Post operative auscultation when so-called "gas-pains" occur demonstrates the recovery from intestinal paresis of surgery. Enemas will be effective, feeding may be started, and prophylactic gastric suction may be discontinued. In the reduction of acute hernia where the viability of the intestine is questionable the presence of peristalsis obviates resection. When a quiet or silent abdomen is present one must suspect the progress of gangrene.

Abdominal auscultation is an important part of physical examination. It should be taught. Constant auscultation of peristalsis is necessary, however, to learn it. As in listening to the heart and lungs, practice is most important.

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